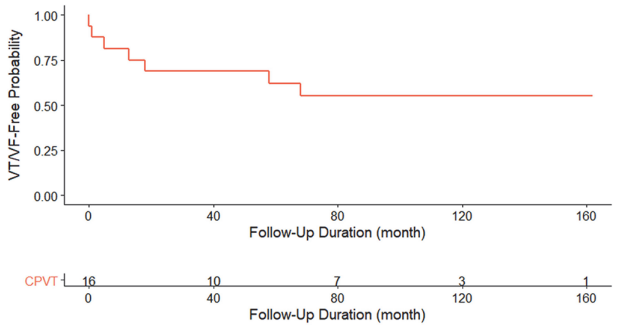
variants have been described else-where (c.14848G>A, c.12475C>A, c.7420A>G, c.11836G>A, c.14159T>C, c.10046C>T and c.7202G>A). c.14861C>G is a novel RyR2 variant that has not been reported outside this cohort. All patients were treated with beta-blockers, three patients received amiodarone and two received verapamil. Sympathectomy (n=8) and implantable-cardioverter defibrillator implantation (n=3) were performed. Over a median follow-up of 127 (IQR: 97–143) months, six patients suffered from incident VT/VF. No significant predictors were identified on Cox regression.

Abstract 104 Table 1 Baseline clinical and demographic characteristics. Categorical and continuous variables were compared between groups using Fisher's exact test or t-test, respectively. Bolded text indicates P<0.05

Variable	All CPVT patients (n=16)	CPVT patients with incident VT/VF on follow-up (n=6)	CPVT patients without incident VT/VF on follow-up (n=10)	P-value
Female	8 (50.0)	4 (66.7)	4 (40.0)	0.302
Presentation Age (years)	10.8±4.4	10.7±4.1	10.8±4.9	0.478
Diagnosis Age (years)	11.4±4.4	11.3±3.8	11.4±5.0	0.489
Presentation to Diagnosis (months)	7.7±10.4	7.5±6.8	7.8±12.4	0.479
Family History of CPVT/SCD	3 (18.8)	3 (50.0)	0 (0)	0.137
Initial syncope	14 (87.5)	5 (83.3)	9 (90.0)	0.696
Initial VT/VF/ SCD	5 (31.3)	1 (16.7)	4 (40.0)	0.330
Initial palpitations	4 (25.0)	3 (50.0)	1 (10.0)	0.074
Initial seizure	6 (37.5)	3 (50.0)	3 (30.0)	0.424
Follow-Up Duration (months)	116.3 ±35.9	131.7±17.5	107.0±41.5	0.904

Abstract 104 Table 2 Baseline electrocardiographic data. Categorical and continuous variables were compared between groups using Fisher's exact test or t-test, respectively. Bolded text indicates P<0.05

Variable	All CPVT patients (n=16)	CPVT patients with incident VT/VF on follow-up (n=6)	CPVT patients without incident VT/VF on follow-up (n=10)	P-value
Heart Rate	81±24	77±19	83±27	0.323
P-Wave Duration	94±18	106±8	85±18	0.881
PR Interval	163±53	191±77	144±14	0.955
QRS Interval	90±25	95±36	85±15	0.757
QT Interval	369±59	391±43	354±66	0.874
QTc Interval	424±34	435±34	417±33	0.836
P Axis	40±35	35±46	44±26	0.338
QRS Axis	67±29	59±32	73±28	0.203
T axis	43±45	62±52	26±32	0.919



Abstract 104 Figure 1 Kaplan-Meier survival curve for catecholaminergic polymorphic ventricular tachycardia (CPVT) patients

Conclusion All CPVT patients from Hong Kong presented at or before 19 years of age. Genetic variants in RyR2 were identified.

Declaration A Preprint of this study has been published on Medrxiv.

Conflict of Interest None

105 PREDICTORS OF APPROPRIATE THERAPIES IN THE CONTEXT OF CARDIAC SARCOIDOSIS AFTER TRANSVENOUS DEFIBRILLATOR IMPLANTATION

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Introduction Sarcoidosis is a multi-systemic inflammatory disorder characterised by non-caseating granulomata. Cardiac sarcoidosis (CS) is typified by the presence of myocardial inflammation and confers adverse prognosis, as it can be associated with conduction abnormalities, congestive heart failure, ventricular arrhythmias (VA) and sudden cardiac death. European Society of Cardiology (ESC) guidelines advocate implantable cardioverter-defibrillator (ICD) implantation with concurrent cardiac resynchronisation therapy as a Class IIa indication in patients with CS that have an indication for permanent pacing with left ventricular ejection fraction (LVEF) <50%.

Purpose Although established guidelines advocate ICD implantation in specific sub-cohorts, there remains a paucity of data on outcomes. We conducted a systematic review of published literature to assess outcomes in patients with CS treated with ICD.

Methods Observational studies of patients with definite or probable CS and ICD implantation were identified from multiple databases from inception to 21st May 2021. Relevant studies were scrutinised for inclusion and data extraction was performed using a pre-specified template. The primary outcome of interest was appropriate ICD therapies with a secondary outcome of all-cause mortality.

Results Eight retrospective, non-randomised studies were identified, comprising 530 patients with follow-up period of 24 to

66 months (weighted average 40 months). Mean age was 53.9 years with average ejection fraction of 41.3%. Overall incidence of appropriate therapy, reported in all studies, was 38.1% during the follow-up period. Left ventricular systolic dysfunction (LVSD) with ejection fraction < 40% was a predictor of appropriate therapy in the majority of studies, as were sustained VA during electrophysiological testing (EP) in one study. All-cause mortality was reported in six studies, with incidence of 6.0% over a median follow-up period of 42 months; only two mortality events were linked to a primary arrhythmic cause.

Conclusions Appropriate ICD therapies in patients with CS is commonly associated with LVSD, which may act as a surrogate for scar burden. The utility of EP testing in this setting remains unclear.

Conflict of Interest No

Heart failure

106 UNCOVERING MECHANISMS OF OBESITY-RELATED HEART FAILURE USING CARDIOVASCULAR MAGNETIC RESONANCE IMAGING IN THE UK BIOBANK

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Introduction Obesity is a rising public health crisis and a major risk factor for heart failure (HF). However, underlying mechanisms are incompletely understood. In this study, we investigate longitudinal associations of obesity with incident HF and cardiovascular imaging phenotypes in the UK Biobank (UKB). Importantly, we use cardiovascular magnetic resonance (CMR) to investigate potential mechanisms driving the obesity-HF relationships.

Methods The UKB cohort comprises over half a million individuals recruited from across the UK between 2006–2010. The UKB imaging study, which includes CMR, commenced in 2015 with plan to scan a random 20% subset of the original cohort. We defined obesity using body mass index (BMI) and waist-to-hip-ratio (WHR) measured at baseline recruitment. Incident HF events were identified through linked Hospital Episode Statistics data (censor date December 2021). CMR scans were analysed using an automated pipeline. We used Cox proportional hazard regression models adjusted for potential confounders to estimate the associations of BMI and WHR with incident HF in the whole sample. We used linear regression to characterise obesity (BMI, WHR) associations with CMR phenotypes. Finally, we used multiple mediation analysis to define the role of obesity-related cardiac remodelling in driving its associations with incident HF, independent of cardiometabolic diseases (diabetes, hypertension, high cholesterol).

Results In 491,606 UK Biobank participants (mean age 56.6 years, 54.3% women) over 12.2±0.9 years of prospective follow-up, higher BMI [HR 1.34 (1.32, 1.37)] and WHR [HR 1.30 (1.30, 1.36)] were associated with a greater hazard of HF. In the subset of participants with CMR (n=31,107), greater obesity was associated with adverse left ventricular (LV) structure (higher LV mass, greater concentricity), poorer LV global functional index, and lower myocardial native T1. In multiple mediation analysis, hypertension had an important role in mediating the associations of obesity with incident HF. Adverse LV remodelling (higher LV mass, greater concentricity) were also major mediators of the obesity-HF associations, independent of cardiometabolic disease. Notably, higher native T1 (indicated greater myocardial fibrosis) mediated a significant fraction of the relationship between obesity and incident HF. Given that in the whole cohort greater obesity was related to lower native T1, this observation may indicate different stages of progression in obesity-related cardiac remodelling.

Conclusion Our findings demonstrate the association of obesity with a greater risk of HF and adverse alterations of LV structure, function, and myocardial character. Importantly, we highlight the role of specific adverse cardiovascular remodelling patterns in driving obesity-HF associations. Thus, our findings highlight the growing public health importance of obesity as a driver of HF and propose novel channels for dedicated mechanistic research.

Conflict of Interest None

107 EMPAGLIFLOZIN IN HEART FAILURE WITH A PRESERVED EJECTION FRACTION ≥50%: RESULTS FROM THE EMPEROR-PRESERVED CLINICAL TRIAL

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Background In EMPEROR-Preserved, empagliflozin reduced the composite primary endpoint of cardiovascular (CV) death/hospitalisation for heart failure (HHF) in patients with heart failure (HF) with left ventricular ejection fraction (LVEF) >40%. We assessed the effect of empagliflozin in patients with a preserved LVEF ≥50% (considered 'true HFpEF' by many clinicians) and contrasted it with HF patients with mildly-reduced LVEF of 41–49% (i.e., <50%).

Methods Of 5,988 randomised patients, 1,983 had LVEF <50% and 4,005 had LVEF ≥50%. The outcomes included (1) the primary endpoint, (2) first and total HHF, (3) change in KCCQ-Clinical Summary Score (CSS), and (4) NYHA class at Week 52.

Results Patients with LVEF ≥50% (vs LVEF <50%) were more frequently women and were older; they had median NT-proBNP 946 pg/mL, and mean eGFR 59 mL/min; approximately half had atrial fibrillation or diabetes at baseline. In patients with LVEF ≥50%, empagliflozin reduced the risk of CV death/HHF by 17% (p=0.024), driven by a reduction in HHF (see table). Time-to-first-event of HHF was reduced by 22% (p=0.013) and total HHF by 17% (p=0.113). Empagliflozin produced meaningful improvements in KCCQ-CSS and NYHA class at Week 52. Compared with placebo, empagliflozin increased KCCQ-CSS in patients with LVEF ≥50% by 1.46 points (0.42–2.51; p=0.006); these empagliflozin-treated patients were 34% more likely to be in a lower NYHA class (p<0.001).